

The Brain Aneurysm Institute

Multidisciplinary Care of Patients with Hemorrhagic and Ischemic Stroke

WINTER 2021

Neurovascular News



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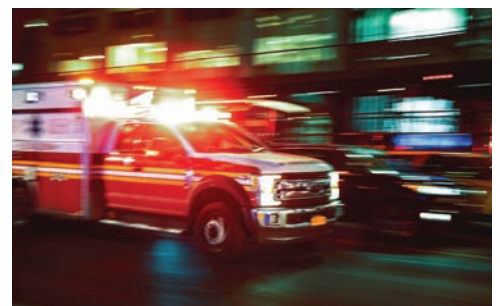
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COVID-19 Acute Ischemic Stroke and Intracranial Hemorrhage

In April 2020 a report appeared from New York City claiming a higher risk of stroke in COVID-19 patients. A survey was conducted in New England Hospitals and we found no increase in the incidence of stroke. In fact, as COVID-19 cases increased in March and April 2020, the actual number of strokes treated throughout New England decreased. This was felt to be due to patient's fear to come to the medical system to seek care for stroke symptoms and therefore not being triaged for stroke care. COVID-19 patients can indeed develop ischemic stroke. This is usually part of an end organ failure type picture where thrombosis and emboli are occurring in multiple organ systems including the kidneys, the heart and the brain. When acute thrombectomy is performed in these patients they often have multi-organ failure.

There does not appear to be an increased incidence of subarachnoid hemorrhage from intracranial aneurysms as relates to COVID-19. Intraparenchymal hemorrhages may occur as a result of an increased incidence of coagulopathy. These patients are managed with algorithms treating the mass-effect of the hematoma with surgery as indicated.

At the BIDMC Brain Aneurysm Institute we continue to treat urgent and emergent situations of stroke and hemorrhage in patients with and without COVID-19. In addition, we will continue regular outpatient evaluations of patients with newly diagnosed neurovascular problems. These evaluations can be performed with telephonic office visits, video office visits or in person as deemed appropriate based on the severity of the disease. Please contact the BIDMC Brain Aneurysm Institute at **617-632-9940** for further guidance or questions regarding patients with neurovascular disease.

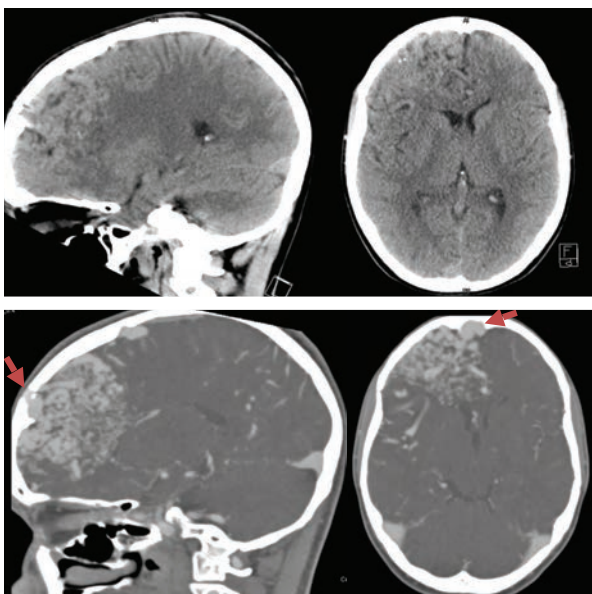


Large Brain Arteriovenous Malformations (AVMs)

Christopher S. Ogilvy, MD, Mirhojjat Khorasanizadeh, MD

An arteriovenous malformation of the brain represents an abnormal shunt of blood from the arterial system to the venous system through a serpiginous tangle of blood vessels. This places the venous circulation under elevated pressure. AVMs may present with seizure, hemorrhage, and headache or may be discovered incidentally. AVMs can occur anywhere throughout the central nervous system and also may occur in other organ systems such as the lung, liver and bowel. Brain AVMs range in size and location. Large AVMs are felt to be lesions larger than 3 cm. Size of the arteriovenous malformation becomes one of the important factors in managing patients with AVMs and may relate to risks of hemorrhage and risks of treatment. Large AVMs are 2.5 times more likely to bleed compared to small lesions¹. While every part of the brain has some level of function, there is certainly those areas that are more “eloquent” than others. Moreover, deep seated AVMs are more likely to bleed compared to superficially located AVMs¹. Therefore, in addition to size, location of the malformation is critical in the decision making as to whether or not to treat an AVM.

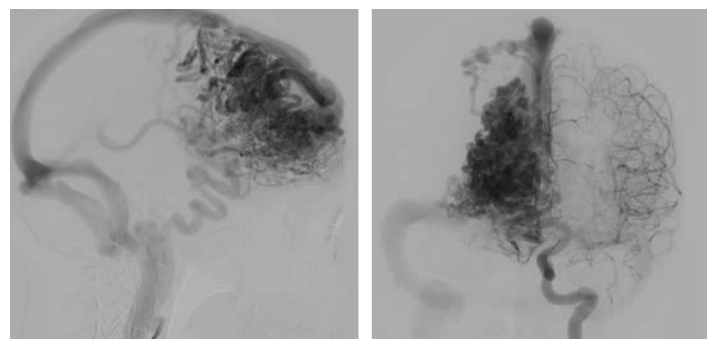
As an example of a large AVM, we present a patient recently managed at the Beth Israel Deaconess Medical Center Brain Aneurysm Institute. The patient was a 25-year-old woman who presented with intense headache. She had no seizure history and no other history of neurologic problems. At the time of presentation, CT scan of the head was obtained with and without – contrast as shown in figure 1.



◀ **Figure 1:** CT scan without (top row) and with (bottom row) contrast. One can see the large area of vascularity in the right frontal region. Note the area of bony erosion where there is a venous varix (arrows).

As is typically appropriate, a cerebral angiogram was obtained to delineate the details and the anatomy of the AVM. It is through such details that decisions can be made regarding the various treatment options available. AVMs may be observed clinically and patients can live with the risk of hemorrhage. This risk is thought to be low per year on (the order of 2 to 4%)². However, this risk is cumulative so the younger the patient, the higher the lifelong risk of hemorrhage. There are certain factors that may modify the natural history risk of an AVM including venous drainage pattern and number of draining veins, presence of venous aneurysms or varices, or arterial aneurysms³. In terms of treatment, lesions may be embolized with a glue-like material through microcatheterizations of the feeding artery. While this is usually used as an adjunct to subsequent surgical resection or treatment with radiosurgery it may well be curative in 10 to 15% of patients⁴. Remaining options for treatment include surgical excision or focused beam radiation called radiosurgery where a high dose of radiation is delivered to the AVM with low dose to the surrounding tissue. The size of the AVM is one of the factors that influence the surgical risk and may also influence the risk of radiosurgical success in that larger lesions are less likely to obliterate than smaller lesions.

A cerebral angiogram was performed on our patient and the large right frontal arteriovenous malformation was found to be fed by arteries from the anterior cerebral artery as well as the middle cerebral artery. Venous drainage occurred through both the deep and superficial venous system of the brain (Figure 2). These features are important in making decisions regarding risk of treatment of AVMs.



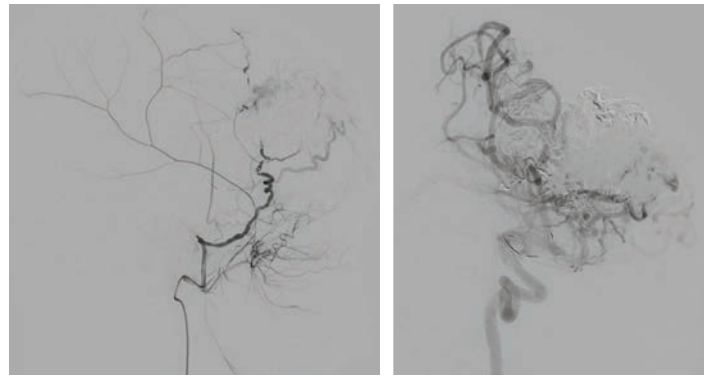
▲ **Figure 2:** Angiogram of the AVM

After careful consideration, it was decided to proceed with staged embolization of the AVM thereby slowly reducing flow through the lesion. We knew this would be only partial treatment and therefore a surgical resection was planned after the final embolization was performed. Five embolization procedures were performed and the final reduction in flow is shown in figure 3. The patient was taken for surgical resection. Surgery for these lesions can be fairly complex and require several hours. In this case, surgery was particularly complicated because there was bony erosion of the skull by a venous varix. This prompted us to perform an innovative procedure which we termed “craniotomy within craniotomy” in order to expose the brain while protecting the varix within the skull from manipulation and hemorrhage (see figure 4). We did a small craniotomy around the eroded area, and another large craniotomy for AVM resection, and temporarily left the varix buried in the bone. Figure 5 shows the final angiogram after surgical resection of the AVM. The patient awoke with a hand that was weak from a supplementary motor area resection of the AVM; however this weakness resolved over the course of 14 days. She was ultimately completely intact from a neurologic standpoint.

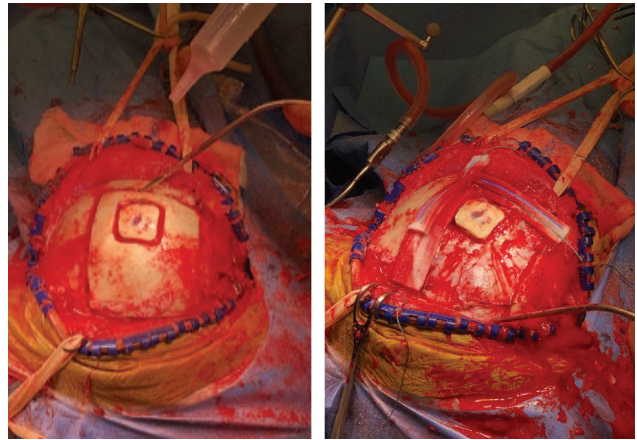
This patient illustrates the value of staged embolization and surgical resection for large brain AVMs. The natural history of this lesion is projected at a 2 to 4% hemorrhage rate and given the patient’s young age this would result in a significant risk of hemorrhage over the remainder of her lifetime. Many hours are spent in the careful planning and performance of the embolization procedures and in the surgical resection. Great teamwork is required with anesthesia, neurology and neurosurgery in the management of these complex patients. However, success can be achieved with AVM resection and resultant reduction in risk of hemorrhage.

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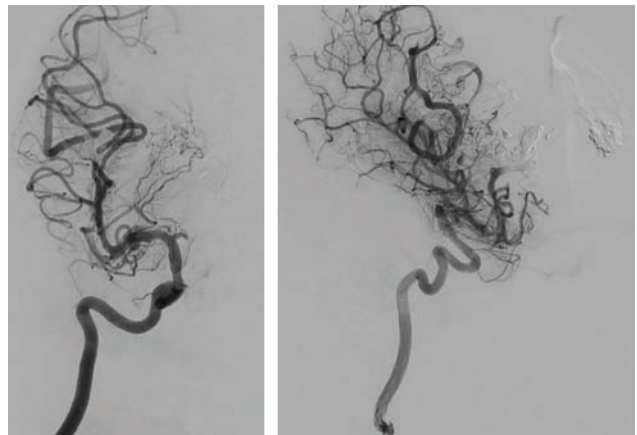
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▲ **Figure 3:** Post embolization angiogram of the AVM



▲ **Figure 4:** “Craniotomy within craniotomy” allowed the surgical team to start resecting the AVM while the bone-eroding varix was temporarily left buried within the skull



▲ **Figure 5:** Postoperative angiogram of the AVM

Subarachnoid Hemorrhage in Setting of Multiple Intracranial Aneurysms – Which one bled?

Dominic A. Harris, MD, Justin Moore, MD, PhD, Christopher S. Ogilvy, MD

Background

Intracranial aneurysms can be found in approximately 2 to 3 percent of the population⁸. Among patients presenting with aneurysmal subarachnoid hemorrhage, multiple intracranial aneurysms are found in 15 to 35 percent of cases⁶. In these cases, it is critical to be able to identify which aneurysm ruptured for treatment planning^{7,9}. Misidentification can have a major impact on the patient's outcome, as rebleeding of untreated ruptured aneurysms significantly increases morbidity and mortality⁴. There are several imaging features that can help determine which aneurysm ruptured, including hemorrhage pattern, aneurysm size, shape, and location. This article provides an overview of how to identify the ruptured aneurysm as well as providing an illustrative case of a patient presenting with subarachnoid hemorrhage and multiple intracranial aneurysms.

Hemorrhage pattern

The hemorrhage pattern on the initial Head CT is the primary indicator of the bleeding source⁸. This is usually determined when focal accumulation of subarachnoid hemorrhage on CT has a predominant laterality or is in proximity to an aneurysm identified on the CT angiogram (CTA). The pattern of subarachnoid hemorrhage has been shown to be a highly accurate way of identifying which aneurysm ruptured⁸. Cases of uncertainty, however, can arise when the hemorrhage is diffuse and symmetric or when there are multiple aneurysms localized near a focal area of hemorrhage^{4,8}. When the hemorrhage pattern is not definitive, determining which aneurysm ruptured can be challenging. This requires one to rely on other features such as size, morphology, or location of the aneurysm⁸.

Aneurysm size and location

Multiple studies have shown size and location to be independent risk factors for aneurysm rupture³. For example, the International Study of Unruptured Intracranial Aneurysms (ISUIA) data suggest that aneurysms in the posterior circulation including posterior communicating artery aneurysms, anterior communicating artery aneurysms, and aneurysms larger than 7 mm have a higher risk of rupture¹⁰. Further studies have confirmed the higher risk of rupture of anterior communicating artery aneurysms specifically among patients with multiple intracranial aneurysms⁷. Nonetheless, aneurysm size is not always a good predictor of rupture. Backes et al., for example, found that in 29 percent of patients presenting

with subarachnoid hemorrhage and multiple intracranial aneurysms, the ruptured aneurysm was not predicted by size³. Therefore, if the larger lesion correlates with the distribution of blood, this is typically the offending lesion. However, there are situations where multiple aneurysms will need to be treated with endovascular or surgical techniques when uncertainty remains.

Aneurysm Morphology

Aneurysm morphology has significant predictive value in determining which aneurysm ruptured in these cases. Irregularly shaped aneurysms have been associated with aneurysm enlargement which portends a higher risk of rupture^{2,7}. Additionally, the presence of a 'daughter' sac has also been associated with higher rupture potential^{1,4,5}. Multiple studies have demonstrated higher mean aspect ratios (height/width) in ruptured aneurysms compared to unruptured aneurysms¹. These morphologic characteristics taken together suggests that irregularity is a surrogate for the aneurysm growth and rupture, which may help with identifying which aneurysm should be treated first in the setting of multiple aneurysms.

Illustrative Case

A 66-year-old female with history of hypertension presented to Beth Israel Deaconess Hospital Emergency Department with severe headache and neck pain. Head CT demonstrated subarachnoid hemorrhage primarily in the right sylvian fissure as well as the suprasellar and perimesencephalic cisterns (Figure 1).

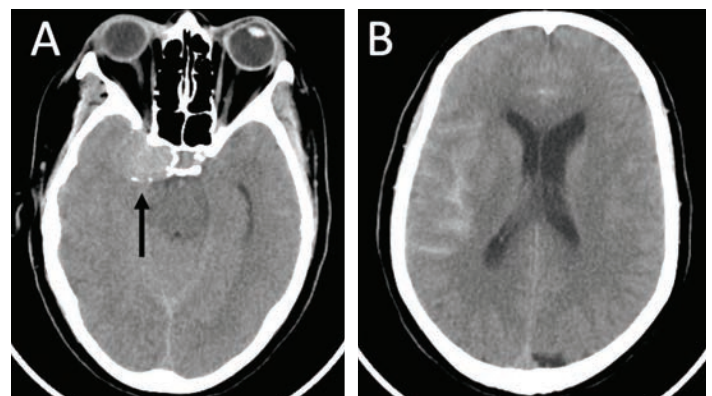


Figure 1: Non-contrast head CT demonstrates subarachnoid hemorrhage primarily in the right sylvian fissure (Fig 1B). Partially thrombosed aneurysm of the right cavernous internal carotid artery is also visualized (Fig 1A).

CT Head Angiogram demonstrated a large cerebral aneurysm involving the distal right internal carotid artery measuring 1.9 x 1.2 cm centered in the region of the cavernous sinus (Figure 2). A smaller lobulated component of the aneurysm was seen extending cephalad near the ICA terminus. The CTA also demonstrated a 4 mm aneurysm in the left ICA terminus. The CTA findings suggested that the large cavernous aneurysm was partially thrombosed, and likely not the source of the subarachnoid hemorrhage given the location and its features.



Figure 2: CT Head angiogram with 3D reconstruction demonstrates three aneurysms. The largest is a right ICA cavernous segment aneurysm (red arrow). Just distal to this is a smaller but highly irregular aneurysm extending cranially into the subarachnoid space (white arrow). On the contralateral side is a saccular ICA terminus aneurysm (yellow arrow).

The patient was taken to the angiography suite where the smaller lobulated component of the right ICA aneurysm was treated with primary coiling. The procedure went well without complication. The patient was monitored in the neuroscience ICU for several days and finally discharged home without further sequela. We plan to treat the larger right cavernous segment ICA aneurysm with flow-diversion as well as the contralateral ICA terminus aneurysm with coiling with or without stent assistance at a later date.

At Beth Israel Deaconess Medical Center Brain Aneurysm Institute, we encounter many patients with multiple intracranial aneurysms. It can often be challenging to identify which aneurysm is the culprit in patients presenting with subarachnoid hemorrhage. Using the combination of hemorrhage pattern and morphologic features of the aneurysm, we make treatment decisions that aim to reduce the risk of rehemorrhage and give our patients the best outcomes.

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Neurovascular Causes of Tinnitus/Bruit

Aristotelis Filippidis, MD, Dominic Harris, MD, Ulas Cikla, MD, Christopher S. Ogilvy, MD

Tinnitus is the perception of noise or “ringing” in the ears. Tinnitus can be differentiated into non-pulsatile versus pulsatile. Non-pulsatile tinnitus usually has its origin in ear pathologies and a referral to an ear-nose-throat specialist (ENT) is needed. In cases of pulsatile tinnitus, the perceived sound by the patient is that of a “whooshing” character, which is synchronized with the patient’s heartbeat. In cases related to a vascular origin, the bruit may be audible to the examining physician as an audible bruit with a stethoscope in the neck or head¹.

Pulsatile tinnitus resulting in a bruit often has a neurovascular cause and in addition to an ENT specialist, a neurovascular referral is often needed. In about 70% of pulsatile tinnitus cases, a cause can be identified with extensive work-up¹⁻³. Vascular causes include arterial or venous vascular pathologies, such as dural arteriovenous fistula (dAVF), arteriovenous malformation (AVM), aneurysm, internal carotid artery stenosis or dissection, congenital vascular variants, transverse sinus stenosis, or increased cardiac output. These pathologies are difficult

to identify without proper cerebrovascular imaging work-up. As a first step we include a computed tomography angiogram (CT angiogram). An MRI and MRA may be helpful as well but a diagnostic cerebral angiogram is often needed as the most sensitive exam for complex cerebrovascular pathologies to clarify the presence of a fistula and its feeding and draining vessels. Non-vascular etiologies of pulsatile tinnitus include neoplasm like paraganglioma, osseous pathology, idiopathic intracranial hypertension, and systemic disorders such as anemia².

Dural Arterio-Venous Malformation (dAVM)/Dural Arterio-Venous Fistula (dAVF)

These lesions are vascular abnormalities in which an arteriovenous shunt is contained within the leaflets of the dura mater, with arterial feeders from the carotid or vertebral arteries penetrating the dura mater. High flow and pressure as well as abnormal communication is present between an artery which shunts directly to a vein. They are usually termed dural arteriovenous fistulas (dAVFs) which is synonymous with dural AVM (dAVM). Most of these lesions (63%) are found at the transverse venous sinus, close to the transverse-sigmoid junction (Figure 1). For intracranial AVMs, only 10-15% are dAVMs, and most are in females with the target patient age group between 40-50s. They are considered acquired lesions with a possible history of trauma or previous thrombosis, resulting in collateral recruitment and revascularization¹⁻⁴.

Presentation includes: 92% pulsatile tinnitus, 89% occipital bruit, 41% headache, 33% visual impairment and 26% papilledema. The symptoms are often related to intracranial venous hypertension. There are varying angiographical presentations with different types but the most important consideration, apart from the annoying tinnitus/bruit, is that fistulas with retrograde cortical venous flow (Venous Hypertension) can lead to intracranial hemorrhage in up to 60% of cases. This is a reason to treat them.

Treatment options, after the patient is seen by a cerebrovascular neurosurgeon and a diagnostic angiogram is obtained, include embolization and possible craniotomy for direct disconnection of the fistula and occasionally stereotactic radiosurgery.

Skull base AVMs

Arteriovenous malformations of the skull base/neck can also present with a bruit/pulsatile tinnitus. An AVM consists of a network of tortuous dilated arteries and veins, referred to as a nidus, through which shunting occurs between arteries and veins without the presence of a normal intervening arteriole-capillary bed. These AVMs may be congenital and can be seen more frequently in younger ages. The lesions recruit vessels mostly from the external carotid artery, or the vertebral artery muscular branches. At our center we have recently encountered such a patient (Figure 2). Treatment options include endovascular embolization in one or multiple sessions,

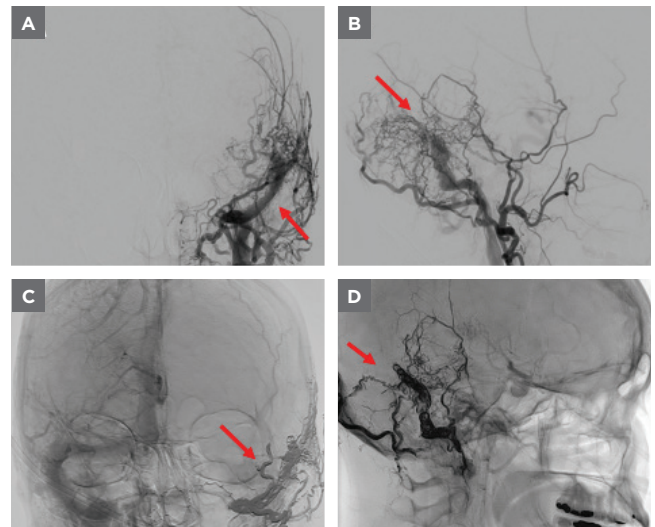


Figure 1: Cognard Type IIa dural AV fistula with contributions from the left occipital, ascending pharyngeal, and middle meningeal arteries. **A:** Left transverse-sigmoid dAVF, AP view, red arrow showing early visualization of venous sinus for this transverse sigmoid dAVF for a patient with left sided bruit. **B:** Lateral view, red arrow shows the fistula, **C:** post embolization treatment result, red arrow shows the part of the dAVF which was embolized with Onyx. **D:** post embolization treatment result, red arrow shows the part of the dAVF which was embolized with Onyx and also coiling was used. A transvenous neck approach was used for the final embolization. The patient is cured with no bruit present any more.

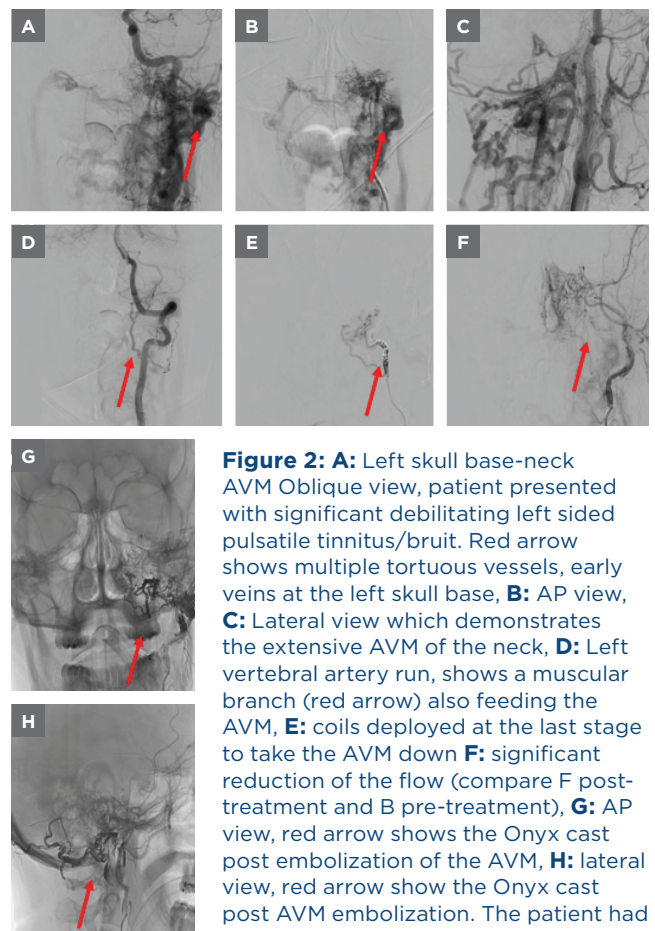


Figure 2: **A:** Left skull base-neck AVM Oblique view, patient presented with significant debilitating left sided pulsatile tinnitus/bruit. Red arrow shows multiple tortuous vessels, early veins at the left skull base, **B:** AP view, **C:** Lateral view which demonstrates the extensive AVM of the neck, **D:** Left vertebral artery run, shows a muscular branch (red arrow) also feeding the AVM, **E:** coils deployed at the last stage to take the AVM down **F:** significant reduction of the flow (compare F post-treatment and B pre-treatment), **G:** AP view, red arrow shows the Onyx cast post embolization of the AVM, **H:** lateral view, red arrow show the Onyx cast post AVM embolization. The patient had a 90% reduction of the tinnitus.

occasionally open surgery and rarely radiosurgery². Such lesions are ideal for collaborative discussions of management. At the BIDMC Brain Aneurysm Institute these patients are presented each week at a multidisciplinary cerebrovascular conference to discuss risks and benefits of each treatment modality.

Petrous apex osseus AVMs are lesions with a bruit. Malik et al. was one of the first to describe intraosseous skull base AVMs⁵. He presented two cases of intraosseous AVMs near the foramen magnum. Both cases were treated surgically or with combined embolization and surgery. Mahmood et al. describe an AVM near the jugular foramen supplied by occipital, ascending pharyngeal and posterior auricular artery draining into internal jugular, epidural and cervical venous plexus⁶. Jung et al. presents 6 cases of intraosseous AVMs⁷. These lesions were associated with osteolytic lesion on CT and dilated venous pouch seen on MR. All lesions were treated via transvenous approach. At times these lesions can present with a temporal hemorrhage as described by Gokce et al⁸.

Skull base tumors

Skull base tumors that cause a neurovascular bruit, must present with significant vascularity and must also present in the vicinity of the petrous apex and inner ear. The most common tumor with these characteristics is a paraganglioma, also known as glomus tumor. This is the most frequent neoplastic cause of pulsatile tinnitus^{1,2,4}. Most paragangliomas are sporadic; about 7-10% are familial and usually autosomal dominant in inheritance. Familial paragangliomas are frequently multicentric (35-50%) and can be associated with multiple endocrine neoplasia (MEN IIa and IIb) or phacomatoses, involving only the jugular bulb (glomus jugulare), the middle ear or mastoid (glomus tympanicum) or both (glomus jugulotympanicum). Most of the paragangliomas located in the temporal bone will present with pulsatile tinnitus. Treatment of these lesions includes tumor embolization, resection or radiation.

Vascular Stenoses

Vascular stenosis is mostly seen in the elderly population as a significant cause of bruit/pulsatile tinnitus. It is thought that a significantly stenosed artery increases vascular flow and causes a bruit. Alternatively, significant stenosis or occlusion may cause a bruit or pulsatile tinnitus on the contralateral side as a result of increased flow. Fibromuscular dysplasia (FMD), described as a segmental non-atheromatous, non-inflammatory vascular disease of unknown etiology may cause pulsatile tinnitus. Often

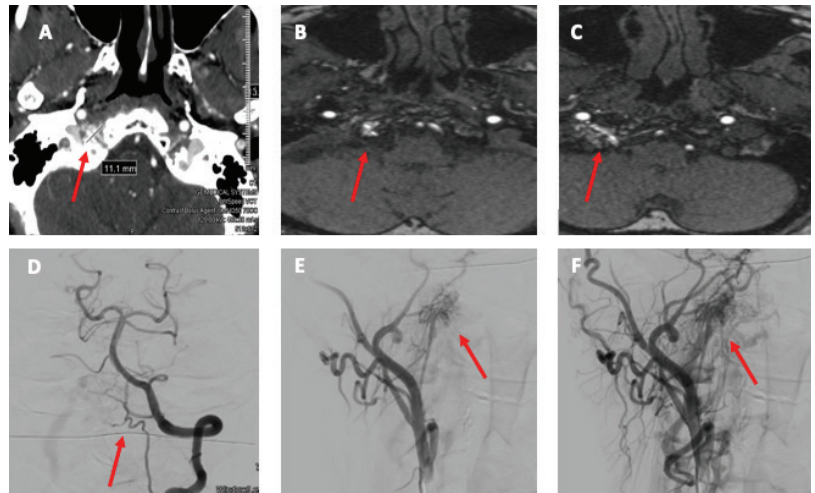


Figure 3: Rare right petrous arteriovenous malformation which is supplied by right external carotid artery branches and muscular branch of the left vertebral artery. **A:** CT angiogram showing the relationship of the AVM with the petrous AVM, **B and C:** MRI time-of-flight sequence identifying the flow voids, **D:** Left vertebral artery run showing a muscular branch contributing to the AVM, **E:** L external carotid run, early and **F:** late phase, with red arrow showing the AVM.

it is a disease of the young leading to vascular stenosis and cerebral ischemia and occasionally bruit. The vessels affected are mostly in the cervical vascular region².

There may be no obvious cause for patients with pulsatile tinnitus. This can be frustrating to deal with for both patient and physician. At the BIDMC Brain Aneurysm Institute we try to work with referring physicians to evaluate patients in such a way as to rule out any potentially dangerous or treatable etiology.

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Save the Date

Ischemic and Hemorrhagic Update: Current Practices and Future Direction

Monday, May 10, 2021

Virtual CME Program

We invite you to join us for the 28th Annual Ischemic and Hemorrhagic Update Course — a yearly comprehensive review of the field of cerebrovascular disease.

The symposium will focus on recent advances in the field of neurovascular disease including current theories on carotid disease, stroke, cerebral hemorrhage, and brain aneurysms and AVMs. We will also cover stroke issues and an overview of hemorrhagic stroke and intraparenchymal hemorrhage.

This year the course will be held virtually and your participation will take place from the comfort and safety of your own home. The Early Bird Rate offers a 10% discount for learners who register up to 6 weeks before the course, March 29, 2021.

<https://cmeregistration.hms.harvard.edu/ischemic2021>

Seminar for Patients and Providers: Moyamoya Disease

In partnership with the Moyamoya Foundation and the Repucci family, the Brain Aneurysm Institute at Beth Israel Deaconess Medical invites you to join us for an educational seminar for healthcare providers and patients on Moyamoya disease.

Thursday, May 6, 2021 | 6–9 p.m.

Fairmont Copley Plaza Hotel, 138 St. James Ave., Boston, MA

To Reserve Your Seat: Please note that we are closely monitoring the COVID-19 pandemic, and our ability to safely gather is dependent on state, as well as Medical Center policies and regulations. We will use this page www.bidmc.org/about-bidmc/events/2021/05/moyamoya-seminar to keep the community up-to-date on our plans should we have to postpone or move to a virtual format.

Please contact Deidre Buckley at 617-632-9713 or e-mail us with any questions dabuckle@bidmc.harvard.edu

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